Echoventriculography in acute myocardial infarction *II: Monitoring of left ventricular performance*

Markku Nieminen and Juhani Heikkilä

From the Cardiovascular Laboratory and the Intensive Care Unit, First Department of Medicine, University Central Hospital, Helsinki, Finland

In acute myocardial infarction the overall left ventricular pump function and the regional performance of the infarcted and non-infarcted myocardial segments were studied serially by echocardiographic techniques in 24 patients during the first week of their illness.

Left ventricular cavity sizes were acutely increased in 62 per cent of the patients ($P < 0.005$). The end-systolic diameter in anterior infarcts increased to the greatest extent, +44 per cent, the end-diastolic diameter by +27 per cent, giving a volume of 246 ± 25 ml. In the anterior myocardial infarcts all the function parameters deteriorated more than in the posteroinferior ones. Ejection fraction was subnormal ($P < 0.005$) in every patient, and mean circumferential fibre shortening (Vcf) was slowed by about 30 per cent ($P < 0.005$).

Regionally, contraction of the infarcted area of the ventricle was asynergic in every instance, and its function was almost totally lost ($P < 0.001$). Systolic paradoxical motion was a constant and stable finding in the anterior infarctions but not so in the posterior ones. While this asynergic systolic contraction may distort echocardiographic measurement of the end-systolic left ventricular volumes, it certainly does not operate at the end-diastolic phase. The serial deviations from normal in the amplitude or velocity of the uninvolved segments were small, but in the case of clearly enlarged end-diastolic volumes these figures in fact indicate supernormal, compensating function. Both overall and regional performance were worst within the first 3 days of infarction, improving thereafter. The patient with a fatal course showed, instead, progressive deterioration.

This noninvasive left ventriculogram by ultrasound gives valuable insight into overall pump function and ventricular volumes, little studied so far in acute infarction, and it may serially quantify the segmental function of both the infarcted and uninvolved regions.

Modern evaluation and management of acute myocardial infarction is increasingly using techniques that allow a quantitative assessment of the left ventricular function. Left ventricular dysfunction certainly is present in most of these patients, more often than is usually recognized from its subtle signs. Since acute infarction may produce a wide range of physiological disturbances, their correct identification and assessment aids in guiding therapy appropriately to prevent development of more serious cardiac failure and to preserve the still ischaemic, but not yet infarcted, myocardium.

Pressure-flow monitoring is now feasible in coronary care units with relatively simple and convenient techniques (Swan *et al.*, 1970). However quantitative assessment of left ventricular volumes,

Received 20 June 1975.

though fundamentally involved with the overall cardiac function, has not yet become established in the acute phase of infarction, as these measurements are usually based on angiographic techniques. We report here our experience in monitoring serially the dimensions of the left ventricle by using quantitative echocardiography in 24 patients with acute myocardial infarction during the first week of their illness. These data were also related to the serial changes found in the regional contraction patterns which were obtained from both the infarcted and noninfarcted left ventricular segments.

Subjects and methods

Twenty-four patients, 20 men and 4 women, with acute myocardial infarction, were studied. They make up that

portion of 30 consecutive patients reported earlier (Heikkilä and Nieminen, 1975) from whom complete serial observations are available. The age range was from 30 to 64 years. The infarction studied was the first one in all the subjects; in 8 patients it was anteriorly and in 16 posteroinferiorly located. The course was uncomplicated in 8 patients, 12 had moderate left heart failure and 4 serious pulmonary oedema and/or shock. All the patients were treated in the intensive care unit.

The ultrasound study on left ventricular function was performed serially on the 1st, 2nd, 3rd, and 7th day from the onset of symptoms with the Picker Echoview II equipment. An echocardiographic technique developed for detailed scanning of the left ventricle ('echoventriculography') was used for evaluation of both the overall and the regional left ventricular function (Heikkilä and Nieminen, 1975; Nieminen, 1975). The various left ventricular regions are recorded by this method using 4 principal ultrasound beam axis directions; several additional sites may be used according to the enlargement of the left ventricle.

The left ventricular end-diastolic and end-systolic diameters were measured in a standard manner (Feigenbaum, 1972) from axis 1 in our echoventriculographic method (Heikkilä and Nieminen, 1975; Nieminen, 1975). Special care was paid to obtain the true transverse axis of the left ventricle, perpendicular to its estimated long axis. The cubed internal diameters were used to calculate left ventricular end-diastolic (LVEDV) and end-systolic (LVESV) volumes (Pombo *et al.*, 1971). Double determinations were used for all measurements. Stroke volume and ejection fraction (EF) were derived from these: $EF = (LVEDV - LVESV) / LVEDV$. Circumferential fibre shortening velocity (Vcf) was modified and measured from the echogram as reported earlier (Nieminen, 1975). The latter study, which described left ventricular overall and segmental functions in 43

healthy subjects, served as normal material for calculations of the statistical significances.

Regional wall motion was measured by the amplitude and the mean velocity of the systolic inward excursion of the particular left ventricular endocardial region under study. These local data were successfully obtained in every serial study from each region of the left ventricle, except for a few early patients in whom the lowest inferior region was not at first systematically scanned (Heikkilä and Nieminen, 1975). The function of at least 8 segments is determined from the basic 4 beam directions, i.e. from the upper and lower halves of the septal, anterior, lateral, and posteroinferior left ventricle segments, with additional detailed recordings from possibly other infarcted regions (see Fig. 9). In addition to their systolic amplitude and velocity, asynergic motion abnormalities were analysed by their area, class (grades: normal, hypokinesia: less than 50% of normal motion, akinesia, and paradoxical motion), and extent, the last combining the changes in area and class together.

Results

Overall left ventricular pump function during first week of infarction

Left ventricular end-diastolic and end-systolic internal transverse diameters, ejection fraction, and modified Vcf for days 1, 2, 3, and 7 after the onset of infarction are displayed in Fig. 1, 2, and 3. Left ventricular internal diameters and volumes were above normal values, $P < 0.005$ for each day. End-systolic diameters ranged from 34 to 60 mm; 62 per cent were above 47 mm (normal mean $+2$ SD), and 86 per cent over about 42 mm (normal mean $+1$ SD) and the end-diastolic ones ranged from 42

FIG. 1 Serial changes in mean left ventricular end-diastolic and end-systolic internal transverse diameters shown by echocardiography in acute myocardial infarction. Both are much larger than normal ($P < 0.005$), the end-systolic one more so, and in anterior infarctions more than in posteroinferior ones.

to 72 mm; 62 per cent over 58 mm (normal mean ± 2 SD), and 76 per cent over 53 mm or normal mean ± 1 SD. In anterior infarctions this cardiac dilatation was most distinct, the mean end-diastolic cavity diameter increased by 27 per cent (61.1 ± 6.5 mm), and the end-systolic one by 44 per cent (52.9 ± 7.0 mm) on day 2. In posterior infarctions, the end-diastolic diameter was most increased by 20 per cent (57.5 ± 7.9 mm) on the 7th day, and the end-systolic one by 31 per cent (47.9 ± 7.1 mm) on the 2nd day. Expressed as end-diastolic volumes, where the cube function of the transverse dimension may be applied with an acceptable accuracy, the left ventricular size in the anterior infarction was 246 ± 25 ml ($+106\%$) and in the posterior ones 208 ± 92 ml ($+78\%$); in the total series, in 75% of the patients it was over 170 ml (normal mean ± 2 SD).

Stroke volumes calculated from the echograms were lowest on the 1st day in anterior infarctions (76 ± 26 ml) and on the 3rd day in posterior infarctions (73 ± 25 ml), the range being from 11 to 127 ml. These values did not differ significantly from normal (62.0 ± 14 ml).

Ejection fraction remained below 50% in all the acutely infarcted hearts. The lowest figures occurred on the 2nd day in anterior ($32 \pm 17\%$) and posterior infarctions ($39 \pm 12\%$), $P < 0.005$ for both. These values improved thereafter but remained subnormal during the whole observation period.

Vcf worsened from the 1st ($P < 0.05$) to 3rd day

FIG. 2 Ejection fractions were uniformly reduced in both anterior and posteroinferior acute myocardial infarctions when assessed by echocardiography.

FIG. 3 Serial changes in modified circumferential fibre shortening velocity (Vcf). About 30 per cent slowing occurs during the first week of acute myocardial infarction.

($P < 0.005$) similarly in anterior and posterior infarction. It was less than 0.32 (mean -2 SD) in 17 per cent, and less than 0.48 (mean -1 SD) in 92 per cent. In anterior ones Vcf continued to decrease further with time, reaching its lowest figure on the 7th day (-38% , 0.40 ± 0.08 circ/s; normal value 0.64 ± 0.16 circ/s), at which time only it differed from the posterior infarctions ($P < 0.05$). In posterior infarctions Vcf did not decrease any further after the 3rd day (-27% to -24% , 0.46 ± 0.07). In contrast to the tendency of ventricular function to improve after the initial days, it deteriorated continuously in the patient who died in hospital.

Sequential changes in regional left ventricular asynergy

Abnormal motion of the infarcted area developed in every patient. The time of maximum asynergic wall motion was the 1st day in 8 patients, 2nd day in 5, and 3rd day in 11, but no later in any of the 24 patients. Serial mean values characterizing abnormal motion of the infarcted left ventricular regions and of the uninvolved segments are displayed in Fig. 4 and 5.

A striking difference exists between anterior and posterior acute myocardial infarctions in regard to segmental function. In anterior infarctions the damaged segment always moved paradoxically and this motion progressed early to its maximum extent, in the majority of patients on the 1st day (in 6 of the 8 patients, and in the other 2 on the following

FIG. 4 Amplitudes of systolic wall motion in infarcted and uninvolved myocardial segments. While motion of the infarcted areas are significantly reduced, or even paradoxical, that of the noninfarcted regions remain mainly within normal ranges. Positive values indicate systolic inward movement and negative values outward movement.

day) and then remained largely unchanged for a week. The mean amplitude of the systolic outward pulsation was 3.2 ± 3.0 mm. In the posterior infarctions a paradoxical pulsation was an exception; nevertheless, a remarkable reduction of wall motion always occurred in the posterior infarcted areas too (Fig. 6). The asynergic changes of the posterior wall developed more slowly: the maximum was seen on the 1st day in 2 patients, on the 2nd day in 5, and on the 3rd day in 9 of the 16 patients with posterior infarctions. When the maximum abnormality was reached on the 3rd day, the mean amplitude of the asynergic wall motion was only 1.9 ± 2.3 mm ($P < 0.001$) and the mean velocity 5.2 ± 7.3 mm/s ($P < 0.001$), i.e. a reduction by 76 per cent and 84 per cent below the normal, respectively. Thereafter the systolic motion at the infarcted posterior regions slightly improved.

The amplitude of the asynergically moving areas was described serially also as increasing, decreasing, or unchanged by comparing the findings between the beginning and the end of the first week. No clear pattern emerged: in 7 patients the contraction disorder improved, in 9 it remained unchanged, and

in 8 the abnormality became worse. However, a difference between anterior and posterior infarctions was again seen. The changes took place mainly in the posterior infarction cases: 6 of the 9 with unaltered amplitude were anterior infarctions, and there were only 2 anterior ones that showed any change: worsening of the paradoxical pulsation after the 1st day.

When serial changes in the qualitative classification of the asynergic motion were studied using the classes paradoxical bulge, akinesia, hypokinesia, or normal motion, no shift from the initial mode was seen in the majority of patients, i.e. in 19 of the 24. In the other 5 the class improved, these had a posterior infarction. Thus the class never worsened in the course of the first week of infarction in either the posterior or the anterior ones. The latter in fact were all already initially in the paradoxical, i.e. the poorest, class.

The extent of the asynergy involving those areas of the left ventricle which showed motion abnormalities was also determined as follows. The abnormality of motion was calculated from 4 anterior (AW1, AW2, AW3, AW4) and 3 posterior (PW1, PW2, PW3, Heikkilä and Nieminen, 1975; Nieminen,

FIG. 5 Systolic wall velocities in infarcted and uninvolved myocardial segments. Because the anterior infarcted segments always moved paradoxically, their systolic velocities are marked as zero. In posteroinferior infarctions the greatest depression of systolic velocity is seen on the 3rd day, with almost total loss of contraction. On the same day the motion of the noninfarcted anterior wall is most active.

1975) regions (see Fig. 9) using both the number of sites showing abnormal motion and the shift of the class of asynergy. This index of asynergic motion decreased in 14 of the 24 patients between the 2nd or 3rd and the 7th day. In 10 of these 14 patients the infarct was a posterior one. In only 2 posterior infarcts was an increase of this asynergy observed, and thus in 4 posterior and 4 anterior infarction asynergy remained unchanged.

Function of uninvolved regions

The amplitude and velocity of the systolic contraction in the left ventricular noninfarcted regions remained mainly within the normal ranges in both the anterior and posterior infarctions (Fig. 4 and 5). In the anterior infarctions the greatest motility of the healthy posterior myocardium was seen during the 1st day, the velocity being increased by +26 per cent (48.3 ± 13.9 mm/s; $P < 0.02$) and the amplitude by +1 per cent (8.4 ± 3.8 mm; NS). These values then fell below normal by the 3rd day, -5 per cent, 36.0 ± 4.3 mm/s (NS) and -16 per cent, 7.0 ± 1.8

mm (NS), respectively, to improve slightly thereafter. In posterior infarctions the amplitude of the healthy anterior wall remained serially stable at the high normal range (+18%, 5.8 ± 1.5 mm; NS) while its velocity rose transiently by +29 per cent (31.7 ± 8.6 mm/s, NS) on the 2nd and 3rd days (Fig. 4, 5, and 6).

Development of electrocardiographic abnormalities of infarction in relation to asynergy

Since the electrocardiographic abnormalities in acute myocardial infarction often develop gradually, and since in this echo study the same was seen to be the case with segmental wall motility too, both were analysed together. Of the 8 anterior infarcts, 6 already showed the most severe grade of asynergy during the 1st day. At this time, however, 4 patients had only ST-T changes of injury in the electrocardiogram and did not develop fresh Q waves until the next day. In the 4 other patients with anterior infarction the already initially present Q waves remained unchanged.

FIG. 6 Left ventricular chamber sizes and regional wall motions seen in echocardiography in posteroinferior and anterior acute myocardial infarction. The uninvolved segments were selected for this panel to show the enhanced function which was frequently seen on the first day. The asynergy in anterior infarctions is characteristically a paradoxical pulsation, while in posteroinferior infarctions hypo- or akinetic modes prevail. (AMI=acute myocardial infarction, AW=anteroseptal wall, ECG=electrocardiogram, LV=left ventricle, LVEDD=left ventricular end-diastolic diameter, LVESD=left ventricular end-systolic diameter, MV=mitral valve, PW=posterior wall of LV, RVW=right ventricular wall.)

In the cases of posterior infarctions 15 echo-electrocardiogram comparisons were available. Asynergic motion at the infarcted area deteriorated slightly in 12 patients during the first 3 days, and worsening was also noted in 6 of these patients electrocardiographically. Initial ST-T changes alone progressed to fresh large Q waves in 4 of these 6, and in 2 the Q waves deepened only. In 5 patients with posterior infarction the infarction electrocardiogram remained unchanged, and in 1 it improved.

Discussion

The following findings emerged from this serial study on left ventricular function by echocardiography. 1) Remarkable alterations take place in both

the overall and the segmental left ventricular performance during the first week of acute myocardial infarction. 2) The echocardiographic picture seen in the anterior acute myocardial infarctions clearly differs from that of the posterior ones (Fig. 7). 3) Asynergic contraction of the left ventricle was documented in all patients with acute infarction by echo left ventriculograms. Such nonuniform contraction may make the volumetric estimations of left ventricular function partially unsatisfactory, so that the ellipsoid approximation may lead to erroneously large, or small, ventricular volume at end-systole. 4) Echo was frequently able to detect distinct abnormalities of ventricular contraction at the time when electrocardiographic changes were still in their initial stage.

Overall left ventricular performance by echo in acute infarction

Left ventricular performance is in a complex way integrated with diastolic ventricular size and pressure (preload), systolic size and pressure (afterload), inotropic state, heart rate, and, especially in ischaemic heart disease, synergy of contraction (Braunwald, 1971). However, in the clinical setting of acute myocardial infarction, only recently have radionuclide cardioangiography and ultrasound provided a means to visualize the heart cavities using a noninvasive technique, with a reasonable quantitative accuracy (Feigenbaum, 1972; Gibson, 1973; Quinones, Gaasch, and Alexander, 1974; Ratshin, Rackley, and Russell, 1974).

Echocardiography in the present study documented various abnormalities in left ventricular function in 100 per cent of the patients, and in the majority of patients (75%) the left ventricular end-diastolic size was significantly enlarged. These findings then confirm the invariable presence of the left ventricular dysfunction which has been found in clinical and haemodynamic studies of the acute phase of infarction (Hamosh and Cohn, 1971; Heikkilä, Luomanmäki, and Pyörälä, 1971a; Gunnar, Loeb, and Rahimtoola, 1974).

The greatest abnormality in the pump function occurred in the end-systolic size and asynergic contraction of the left ventricle. This is obviously

the result of the inherent pathophysiological alteration of the infarction: the regional loss of contractile element function (Swan *et al.*, 1972). The great significance of the systolic distortion in the asynergic segments in further stealing pump performance has been convincingly shown by Parmley *et al.* (1973). The contractile force of the uninvolved segments is largely dissipated in generating tension so that inadequate capacity is left for the ejection of blood from the ventricle. The present figures also indicate that asynergic infarcted zones mainly exceeded the critical size of 25 to 30 per cent of the left ventricle, because the size of asynergy correlates well both with the end-diastolic volume and with the ejection fraction (Herman and Gorlin, 1969; Feild *et al.*, 1972; Hamilton, Murray, and Kennedy, 1972; Kitamura *et al.*, 1973; Parmley *et al.*, 1973; Rigo *et al.*, 1974). The time of poorest overall pump function on the 2nd and 3rd day coincides with an increased compliance of infarcted muscle reported at this time, while the later stiffening supports better overall ventricular performance (Hood *et al.*, 1970; Forrester *et al.*, 1972).

It is remarkable how much also the end-diastolic size of the left ventricle increased acutely, especially with the anterior infarctions. These data in man agree with those in experimental infarction which have indicated an immediate utilization of the Frank-Starling compensation mechanism in main-

SUMMARY OF OVERALL AND REGIONAL LV FUNCTION BY ECHO IN POSTERIOR AND ANTERIOR ACUTE INFARCTIONS

FIG. 7 Average ventricular diameters and systolic motion of both the infarcted and the uninvolved segments are drawn for a normal ventricle and for posteroinferior and anterior acute myocardial infarctions (2nd day). Pump performance is poorest in the anterior infarctions, where the largest ventricular volumes with the lowest ejection fractions are seen, also with the greatest regional asynergic changes. Uninvolved segments show hyperfunction if considered in relation to chamber volume. The dotted lines represent overactive regional function often seen particularly in the uncomplicated infarctions.

taining the normal stroke volume (Harley, Behar, and McIntosh, 1968; Heikkilä, Tabakin, and Hugenholtz, 1972). Among chronic heart diseases the highest preload stresses notably occur in subjects with coronary heart disease associated with segmental left ventricular asynergy (Ratshin *et al.*, 1974): such findings likewise reflect the grave nature of the postinfarction left ventricular dysfunction.

Echocardiographic studies of left ventricular end-diastolic or stroke volumes in acute infarction have so far been reported from a few small series of patients. Broder and Cohn (1972) stated that the left ventricular diameters remained normal in 5 patients despite high filling pressures of 28 to 36 mmHg (3.5 to 4.8 kPa), but Cahill *et al.* (1973) described in 17 patients increases of end-diastolic volume and decreases of ejection fraction which correlated with the clinical severity of acute infarction. In their study the end-diastolic volumes in the NYHA groups 3 and 4 were of the same magnitude (232 ml) as in the patients with anterior infarction in our series (246 ± 25 ml). Stroke volumes by echocardiography have been of the same order as in our study (Pombo *et al.*, 1971; Ratshin, Rackley, and Russell, 1972; Smith *et al.*, 1974; Chapelle *et al.*, 1974).

The distinct acute changes in the left ventricular

size in the present echocardiographic study agree excellently with the picture of acute myocardial infarction which was described in 38 patients evaluated within 48 hours from the onset of symptoms by Rigo *et al.* (1974) by biplane gated scintiphotography. The left ventricular end-diastolic volume was increased by +57 per cent in their heart failure group and by +118 per cent in the cardiogenic shock group, corresponding to the figures in the present series of +78 per cent, 208 ± 92 ml, in the posterior, and +106 per cent, 246 ± 25 ml, in the anterior infarctions. In their study the end-systolic volume was calculated from the biplane areas providing the best accuracy: the remarkable increases were +146 per cent and +320 per cent respectively, and the mean ejection fraction was 36 per cent. Here the increase in the anterior infarctions would result in figures of +230 per cent (168 ± 58 ml), though the calculation of end-systolic volume from a single echo axis is in contrast to the end-diastolic volume, unsatisfactory because of systolic asynergy as discussed later. Another scintigraphic study also showed a similar high prevalence of abnormal volumes as was obtained here by echocardiography (Kostuk *et al.*, 1973).

Echocardiographic left ventricular function in anterior and posterior infarction

Greater depression of left ventricular pump function was evident by echocardiography in anterior myocardial infarctions than in posteroinferior ones. This finding concurs with the haemodynamic difference documented between anterior and posterior infarctions (Russell, Hunt, and Rackley, 1973). Regional function also was poorer in anterior infarctions: asynergy manifested as persistent paradoxical pulsation, as compared with less stable akinesia or hypokinesia in posterior ones, and the uninvolved myocardial regions tended to move less vigorously. These abnormalities may have been slightly modified by the superimposed recoil of the left ventricle as a whole. But the latter is a small and late systolic event (McDonald, Feigenbaum, and Chang, 1972), in contrast to what was seen in our patients when paradoxical motion was already maximal in the isovolumic phase, and similar to the findings noted in experimental infarction (Heikkilä *et al.*, 1972).

Serial changes in overall and regional left ventricular function

The serial monitoring of the left ventricular pump function was limited here to one week. Within this time the pump performance was poorest on the 2nd and 3rd days by all echo parameters. This

FIG. 8 Echocardiographic monitoring of a progressive power failure. In this 34-year-old man the noninfarcted myocardium failed to compensate for an extensive and distinctly paradoxically moving anterior myocardial infarction. Ventricular volume became doubled and ejection fraction fell to one-third of the normal before death. Necropsy findings were displayed in Fig. 8 of our earlier report; Heikkilä and Nieminen (1975).

course agrees with the results obtained with ultrasound (Wharton, Smithen, and Sowton, 1971), and with other methods (Thomas, Malmcrona, and Shillingford, 1965; Hood *et al.*, 1970; Heikkilä, Luomanmäki, and Pyörälä, 1971b; Rahimtoola *et al.*, 1972; Kostuk *et al.*, 1973; Gunnar *et al.*, 1974). Regionally, the serial deviations of the noninfarcted segments remained insignificant, remaining in the high normal range in the posterior infarctions, and falling to the low normal level in the anterior infarctions. However, here it should be recognized that even these normal values indicate enhanced segmental function in the presence of an enlarged left ventricle and high wall stress (Heikkilä *et al.*, 1972).

So far the function of the uninvolved myocardial segments has been estimated indirectly (Feild *et al.* 1972; Kitamura *et al.*, 1973; Rigo *et al.*, 1974). In echocardiography, regional changes in both the asynergic and the uninvolved segments may be monitored directly and serially. In clinical decision making, poor contractility of the uninvolved muscle segments detected in association with large acute infarction has, in our experience, been rewarding, as in these cases the clinical course has always been fatal despite aggressive treatment (Fig. 8).

Influence of asynergic contraction on echocardiographic measurements

Asynergically contracting segments have been of great concern when one has tried to evaluate ventricular function quantitatively by echocardiographic dimensional methods which are based on uniformity of the chamber shape (Paraskos *et al.*, 1971; Gibson, 1973; Ludbrook *et al.*, 1973; Popp *et al.*, 1973). These aspects naturally become relevant to the present study, where echocardiography demonstrated asynergic contraction in 100 per cent of the patients.

Theoretically, even large asynergic segments would not have much influence on volume calculations in diastole because nonuniform ventricular geometry appears only during systolic contraction. Derivation of the end-diastolic volumes by the echo diameter should thus be correct even in the acute phase of myocardial infarction because any local aneurysmal bulge which distorts diastolic shape of the ventricle takes weeks to develop. In chronic coronary heart disease, Sweet *et al.* (1973) similarly noted that asynergy mainly influenced the accuracy of calculating end-systolic and not end-diastolic left ventricular volumes.

Left ventricular end-systolic volume is more

FIG. 9 Diagrammatic cross-sections of left ventricle, to show distortion of volumetric analysis caused by asynergy. End-systolic volume varies depending on whether or not the transverse axis transverses asynergic (axis B) or noninfarcted regions (axis A). Since ventricular asynergy is systolic dynamic alteration, this is thought to have little effect on the symmetry of diastolic dimensions, at least in the early phases after myocardial infarction. The three-dimensional schema of the left ventricle shows the 4 major echobeam directions used in the echoventriculography method.

subject to error because in asynergic systole one transverse axis measurement, or even a single plane angiographic silhouette (Vogel, Cornish, and McFadden, 1973), is no longer representative of the entire chamber. Whether the echo dimension traverses an asynergic segment or not, over- or underestimation of end-systolic volume results from the cube function (Fig. 9). In posterior infarction the methodological error will be in the direction of too large an end-systolic volume (axis B in Fig. 9). In contrast, in anterior infarctions (axis A in Fig. 9) the figure will most often be too low, particularly in large anterolateral infarctions not involving the septum. This was the case in 50 per cent of the anterior infarctions in this series. The shape distortion obviously applies also to the calculation of derived parameters of pump performance like ejection fraction and Vcf. However, when used serially, relative changes of these parameters may also be used in monitoring directional changes in the pump performance (Fig. 8).

The above differences are further related quantitatively, not only to the presence of, but also to the size and type of, motion of the asynergic segment. The size may vary greatly from 10 to 70 per cent of the left ventricular mass (Harnarayan *et al.*, 1970; Feild *et al.*, 1972; Kitamura *et al.*, 1973; Rigo *et al.*, 1974). Echocardiographically, the asynergic area is larger in anterior infarctions (Heikkilä and Nieminen, 1975), and also shows more paradoxical systolic outward motion, with corresponding influence on assessment of the actual end-systolic cavity size (Fig. 4 and 6).

When related to ventricular enlargement, one may calculate a theoretical sum of inward movement of the septum and posterior wall to represent a normal stroke volume of, for instance, 60 ml. For a normal-sized ventricle (LVEDD 50 mm) this figure is 10–11 mm; for a 200 ml end-diastolic volume (LVEDD 59 mm) seen here in posterior infarctions, 7 mm; and for 250 ml (LVEDD 63 mm) of the anterior infarctions, 5.5 mm. Deviation from such a 'normalized' sum of the amplitudes of wall motions (Fig. 7) suggests non-representative muscle areas having been traversed by echobeam, functional mitral incompetence, or both.

This work was supported in part by the Paulo Foundation. We are indebted to Miss Virva Markelin for her help in the preparation of this report.

References

- Braunwald, E. (1971). Control of myocardial oxygen consumption. Physiologic and clinical considerations. *American Journal of Cardiology*, **27**, 416.
- Broder, M. I., and Cohn, J. N. (1972). Evolution of abnormalities in left ventricular function after acute myocardial infarction. *Circulation*, **46**, 731.
- Cahill, N. S., Al-Sadir, J., Brooks, H. L., and Resnekov, L. (1973). Left ventricular echocardiograms as a non-invasive functional index following acute myocardial infarction. *American Journal of Cardiology*, **31**, 124.
- Chapelle, M., Benaim, R., Senikies, A., and Chiche, P. (1974). Évaluation de la fonction contractile du myocarde par échographie, au stade initial de l'infarctus du myocarde. *Coeur et Médecine Interne*, **13**, 21.
- Feigenbaum, H. (1972). *Echocardiography*. Lea and Febiger, Philadelphia.
- Feild, B. J., Russell, R. O., Jr., Dowling, J. T., and Rackley, C. E. (1972). Regional left ventricular performance in the year following myocardial infarction. *Circulation*, **46**, 679.
- Forrester, J. S., Diamond, G., Parmley, W. W., and Swan, H. J. C. (1972). Early increase in left ventricular compliance after myocardial infarction. *Journal of Clinical Investigation*, **51**, 598.
- Gibson, D. G. (1973). Estimation of left ventricular size by echocardiography. *British Heart Journal*, **35**, 128.
- Gunnar, R. M., Loebe, H. S., and Rahimtoola, S. H. (1974). *Shock in Myocardial Infarction*, p. 65. Grune and Stratton, New York.
- Hamilton, G. W., Murray, J. A., and Kennedy, J. W. (1972). Quantitative angiography in ischemic heart disease. *Circulation*, **45**, 1065.
- Hamosh, P., and Cohn, J. N. (1971). Left ventricular function in acute myocardial infarction. *Journal of Clinical Investigation*, **50**, 523.
- Harley, A., Behar, V. S., and McIntosh, H. D. (1968). Immediate hemodynamic effects of acute coronary occlusion and their modification by anesthesia. *American Journal of Cardiology*, **22**, 559.
- Harnarayan, C., Bennett, M. A., Pentecost, B. L., and Brewer, D. B. (1970). Quantitative study of infarcted myocardium in cardiogenic shock. *British Heart Journal*, **32**, 728.
- Heikkilä, J., Luomanmäki, K., and Pyörälä, K. (1971a). Serial observations on left ventricular dysfunction in acute myocardial infarction. I. Gallop sounds, ventricular asynergy and radiological signs. *Acta Medica Scandinavica*, **190**, 89.
- Heikkilä, J., Luomanmäki, K., and Pyörälä, K. (1971b). Serial observations on left ventricular dysfunction in acute myocardial infarction. II. Systolic time intervals in power failure. *Circulation*, **44**, 343.
- Heikkilä, J., and Nieminen, M. (1975). Echocardiographic detection, localization and quantification of left ventricular asynergy in acute myocardial infarction. *British Heart Journal*, **37**, 46.
- Heikkilä, J., Tabakin, B. S., and Hugenholtz, P. G. (1972). Quantification of function in normal and infarcted regions of the left ventricle. *Cardiovascular Research*, **6**, 516.
- Herman, M. V., and Gorlin, R. (1969). Implications of left ventricular asynergy. *American Journal of Cardiology*, **23**, 538.
- Hood, W. B., Jr., Bianco, J. A., Kumar, R., and Whiting, R. B. (1970). Experimental myocardial infarction: IV. Reduction of left ventricular compliance in the healing phase. *Journal of Clinical Investigation*, **49**, 1316.
- Kitamura, S., Kay, J. H., Krohn, B. G., Magidson, O., and Dunne, E. F. (1973). Geometric and functional abnormalities of the left ventricle with a chronic localized non-contractile area. *American Journal of Cardiology*, **31**, 701.
- Kostuk, W. J., Ehsani, A., Karliner, J. S., Ashburn, W. L., Peterson, K. L., Ross, J., and Sobel, B. E. (1973). Left ventricular performance after myocardial infarction assessed by radioisotope angiography. *Circulation*, **47**, 242.

- Ludbrook, P., Karliner, J. S., Peterson, K., Leopold, G., and O'Rourke, R. A. (1973). Comparison of ultrasound and cineangiographic measurements of left ventricular performance in patients with and without wall motion abnormalities. *British Heart Journal*, **35**, 1026.
- McDonald, I. G., Feigenbaum, H., and Chang, S. (1972). Analysis of left ventricular wall motion by reflected ultrasound. Application to assessment of myocardial function. *Circulation*, **46**, 14.
- Nieminen, M. S. (1975). Normal left echocardiography. *Annals of Clinical Research*, **7**, 1.
- Paraskos, J. A., Grossman, W., Saltz, S., Dalen, J. E., and Dexter, L. (1971). A noninvasive technique for the determination of velocity of circumferential fiber shortening in man. *Circulation Research*, **29**, 610.
- Parmley, W. W., Chuck, L., Kivowitz, C., Matloff, J. M., and Swan, H. J. C. (1973). In vitro length-tension relations of human ventricular aneurysms. Relation of stiffness to mechanical disadvantage. *American Journal of Cardiology*, **32**, 889.
- Pombo, J. F., Russell, R. O., Jr., Rackley, C. E., and Foster, G. L. (1971). Comparison of stroke volume and cardiac output determination by ultrasound and dye dilution in acute myocardial infarction. *American Journal of Cardiology*, **27**, 630.
- Popp, R. L., Alderman, E. L., Brown, O. R., and Harrison, D. C. (1973). Sources of error in calculation of left ventricular volumes by echography. *American Journal of Cardiology*, **31**, 152.
- Quinones, M. A., Gaasch, W. H., and Alexander, J. K. (1974). Echocardiographic assessment of left ventricular function. With special reference to normalized velocities. *Circulation*, **50**, 42.
- Rahimtoola, S. H., DiGilio, M. M., Ehsani, A., Loeb, H. S., Rosen, K. M., and Gunnar, R. M. (1972). Changes in left ventricular performance from early after acute myocardial infarction to the convalescent phase. *Circulation*, **46**, 770.
- Ratshin, R. A., Rackley, C. E., and Russell, R. O., Jr. (1972). Serial evaluation of left ventricular volumes and posterior wall movement in the acute phase of myocardial infarction using diagnostic ultrasound. *American Journal of Cardiology*, **29**, 286.
- Ratshin, R. A., Rackley, C. E., and Russell, R. O., Jr. (1974). Determination of left ventricular preload and afterload by quantitative echocardiography in man. *Circulation Research*, **34**, 711.
- Rigo, P., Murray, M., Strauss, H. W., Taylor, D., Kelly, D., Weisfeldt, M., and Pitt, B. (1974). Left ventricular function in acute myocardial infarction evaluated by gated scintiphotography. *Circulation*, **50**, 678.
- Russell, R. O., Jr., Hunt, D., and Rackley, C. E. (1973). Left ventricular hemodynamics in anterior and inferior myocardial infarction. *American Journal of Cardiology*, **32**, 8.
- Smith, M., Ratshin, R. A., Harrell, F. E., Jr., Russell, R. O., Jr., and Rackley, C. E. (1974). Early sequential changes in left ventricular dimensions and filling pressure in patients after myocardial infarction. *American Journal of Cardiology*, **33**, 363.
- Swan, H. J. C., Forrester, J. S., Diamond, G., Chatterjee, K., and Parmley, W. W. (1972). Hemodynamic spectrum of myocardial infarction and cardiogenic shock. A conceptual model. *Circulation*, **45**, 1097.
- Swan, H. J. C., Ganz, W., Forrester, J., Marcus, H., Diamond, G., and Chonette, D. (1970). Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. *New England Journal of Medicine*, **283**, 447.
- Sweet, R. L., Russell, R. O., Jr., Moraski, R. E., and Rackley, C. E. (1973). Comparison of left ventricular volumes obtained by biplane angiography and echocardiography in patients with abnormally contracting segments. *Circulation*, **48**, Suppl. 4, 116.
- Thomas, M., Malmcrona, R., and Shillingford, J. (1965). Hemodynamic changes in patients with acute myocardial infarction. *Circulation*, **31**, 811.
- Vogel, J. H. K., Cornish, D., and McFadden, R. B. (1973). Underestimation of ejection fraction with singleplane angiography in coronary artery disease: role of biplane angiography. *Chest*, **64**, 217.
- Wharton, C. F. P., Smithen, C. S., and Sowton, E. (1971). Changes in left ventricular wall movement after acute myocardial infarction measured by reflected ultrasound. *British Medical Journal*, **4**, 75.

Requests for reprints to Dr. Juhani Heikkilä, Cardiovascular Laboratory, University Central Hospital, SF-00290 Helsinki 29, Finland.